

Does Occipital Alpha Neurofeedback Require Critical Branching to Causally Influence LTRCs and Deterministic ERPs?

Tom Campbell

*Institute for Behavioural Sciences, University of Helsinki,
00014 Helsinki, Finland.
tom.campbell@helsinki.fi*

Abstract: Critical branching is a form of theoretical interaction between simple units, which, in the case of the human brain are assumed to be neuronal elements. Zhigalov, Kaplan, and Palva (*Clin Neurophysiol* 2016;127:2882–89) revealed that neurofeedback flash stimulation phase-locked to high-amplitude occipital alpha influences the stimulus-locked occipital alpha-band averages and the power scaling of long-range temporal correlations of alpha-band amplitude fluctuations. At first, it might seem that neurofeedback influenced critical branching alongside there being an interaction between ongoing neuronal activity and evoked responses. However, the causal relations between these neuronal Long Range Temporal Correlations, sustained attention, and any avalanche dynamics are called into question, as is Zhigalov *et al.*'s assertion that the neurofeedback flashes are unconsciously processed rather than capturing attention. Uncorrected concerns about false discovery rate and an objective mathematical error in the cited foundational work for Zhigalov *et al.*'s study are also raised. Without critical branching, an alternative set of illustrative mathematical principles offered a preliminary fit to the effects in the data, whereby neurofeedback influenced the deterministic contribution to single-trial flash-evoked event-related potentials separately from the neurofeedback flash-evoked gain in the amplitude of those oscillations. Accordingly, micro-behaviours produced by the model's separate principles of neurofeedback-related exponential occipital oscillatory alpha gain and deterministic event-related potential generation have macroscale consequences causally influencing the power-scaling of long-range temporal correlations.

Subjects: Neurons and Cognition (q-bio.NC); Adaptation and Self-Organizing Systems (nlin.AO); Biological Physics (physics.bio-ph). **Keywords:** Closed-loop stimulation; deterministic Event-Related Potentials (ERPs); electroencephalography (EEG); critical neuronal dynamics; long-range temporal correlations (LTRCs); mathematical model.

1. Does Occipital Alpha Neurofeedback Require Critical Branching to Causally Influence LRTCs and Deterministic ERPs?

1.1. Introduction

In a neurofeedback investigation, [Zhigalov et al. \(2016\)](#) refer to criticality analyses alluding to self-organised criticality – a theory inconclusively supported by the neuroscientific data. This term self-organised criticality, or just criticality, is often misconstrued as a property of a physical system, whereas self-organised criticality is, rather, a theory of the interactions within multiple simple elements in a physical system. This theory assumes systems of such elements tend to criticality: Inter-elemental interactions, termed critical branching, poise that system in a critical state between responding in two ways. In a critical state, a system can respond either through linear local interactions or through a higher-amplitude nonlinear propagation of the response via a widespread network of elements. That nature of interaction between elements leading to such a system, poised in what is called a critical state, is termed critical branching.

This theory originated in the peer-reviewed articles on systems such as accumulating piles of long-grain rice self-organising into a critical state. In turn the theory become widely popularised across several domains, including systems neuroscience and now clinical neurophysiology. Characteristics of a system in a critical state derived by criticality analyses include $1/f$ spectra, avalanche dynamics, and fractal self-similarity in the scale-invariance of power-law scaling of long-range temporal correlations (LRTCs).

1.2. Fluctuations of sustained attention, LRTCs and avalanche dynamics

Poignant is Zhigalov et al.'s departure from linking a criticality interpretation of neuronal LRTCs to arguably conscious phenomena such as fluctuations of sustained attention: Previously, analysing MEG-derived time-series, [Palva et al. \(2013\)](#) not only identified such neuronal LRTCs, but also an avalanche dynamics – another characteristic of criticality. Investigating power-scaling of LRTCs, during a stimulus detection task, neuronal exponents correlated positively not only with behavioural exponents but also with neuronal exponents at rest. These behavioural LRTCs concern periods of attention when the participant correctly identifies a stimulus at threshold punctuated by periods of inattention when the participant is incorrect. Thus power-scaling of those LRTCs shows that the extent of fluctuation on one timescale determines that on others – psychologically, there is a power-scaling of fluctuations of sustained attention. Measures of the amplitude and duration of highly nonlinear neuronal avalanches during the task correlate negatively with both behavioural and neuronal LRTC exponents. Nevertheless, those avalanche measures did not significantly predict behaviour independently from neuronal exponents ([Palva et al., 2013](#)). These findings call into question the role of avalanches as a cause for transitions between attention and inattention – there is no such avalanche (Private communication, Alexander Y. Zhigalov, 2015). Thus self-organised criticality, encompassing an avalanche dynamics, cannot drive fluctuations of sustained attention.

1.3. Concerns with the motivating study's analyses

Concerns include the objectively incorrect mathematical expression of the parcellation strategy computation (Private communication, Muriel Lobier, 2015). That is, there was a confusion in the in-house data pipeline of Palva and colleagues between the real and imaginary parts of complex numbers used in calculating similarities of phase between time series in measures such as the phase-locking value (PLV) and the distinct imaginary PLV ([Siebenhühner et al., 2016](#); [Korhonen et al., 2014](#)). This confusion has consequences for fidelity and infidelity measures ([Korhonen et al., 2014](#)) used in

the parcellation strategy of the foundational work (Palva *et al.*, 2013); Private communication, Muriel Lobier, 2015) that Zhigalov *et al.* (2016) cite as motivating their study. While this objective error might be trivialised (Private communication, Jaakko M. Palva, 2015), if further weight is to be placed on the results of Palva *et al.* (2013) for the motivation and interpretation of new investigations, errata are warranted demonstrating this concern does not considerably alter those results. A further concern that could also necessitate an erratum to the foundational work for Zhigalov *et al.* (2016)'s study is the elevated false discovery rate of the in-house data pipeline (Private communication, Hugo Eyherabide, 2015) used in the cited foundational work of Palva *et al.* (2013).

1.4. Neurofeedback influences power-scaling of LRTCs without a reported avalanche dynamics

As an advance on this precedent, Zhigalov *et al.*'s eyes-closed rest procedure demonstrates alterations to LRTCs of scalp-measured EEG alpha-band time-series by a flash 12.5 msec after alpha oscillations' peaks exceed a certain value (Fig. 1A). In contrast to Palva *et al.*, Zhigalov *et al.* do not present an avalanche dynamics that would occur if neurofeedback influenced critical branching. If a subset of the hallmarks of self-organised criticality need be present for critical branching to be operational, then surely the conceptual integrity of the notion is subject to question: A critical branching with an avalanche dynamics (Palva *et al.*, 2013) would be theoretically distinct from that seemingly without (Zhigalov *et al.*, 2016).

1.5. ERPs and consciousness

Zhigalov *et al.* claim the neurofeedback to be unconscious. In the auditory domain, ERP componentry such as the auditory mismatch negativity (Näätänen and Winkler, 1999; Campbell *et al.*, 2007; Ruby *et al.*, 2007; Morlet and Fischer, 2014) is generated in a manner classically considered unconscious and unmodulated by attention (for recent debate, see Campbell, 2015; Wiens *et al.*, 2016). Nevertheless, auditory onsets of which participants are unconscious due to physical characteristics of the stimulation do not elicit the N1 and P2 deflections of auditory ERPs (Lightfoot and Kennedy, 2006). Do onsets of visual stimuli that are subliminal due to characteristics of the stimulation have the capacity to elicit ERPs unconsciously? Flashes at a 50% luminance detection threshold elicit ERPs but only on trials when detected (Busch *et al.*, 2009): Trials on which participants performance revealed they were not subjectively conscious of a flash did not elicit an ERP. Zhigalov *et al.* reveal alpha-band ERPs, which are influenced by neurofeedback, because participants are visually conscious of the flashes seen: That is, the experimenter adjusted the luminance so that participants were aware of the flashes even with closed eyes through their eyelids. On balance, in complex paradigms it is possible for stimuli to elicit visual ERP componentry (e. g., Lyrya *et al.*, 2010; Sysoeva *et al.*, 2015) even without conscious awareness of the content of the stimulus that is otherwise supra-threshold in isolation. However, it is hard to imagine how the paradigm of Zhigalov *et al.* (2016) engendered participants to be unconscious of flashes that they could see. No neurofeedback-related changes in the visibility of the flashes through the participants eyelids are reported. The point is that physical characteristics of a stimulus need to be in principle accessible to perceptual consciousness to elicit ERPs. Luminance increments arguably summon attention when a luminance increment defines the to-be-attended stimulation (Yantis and Hillstrom, 1994). Accordingly, consciously perceived luminance increments involuntarily attentional capture.

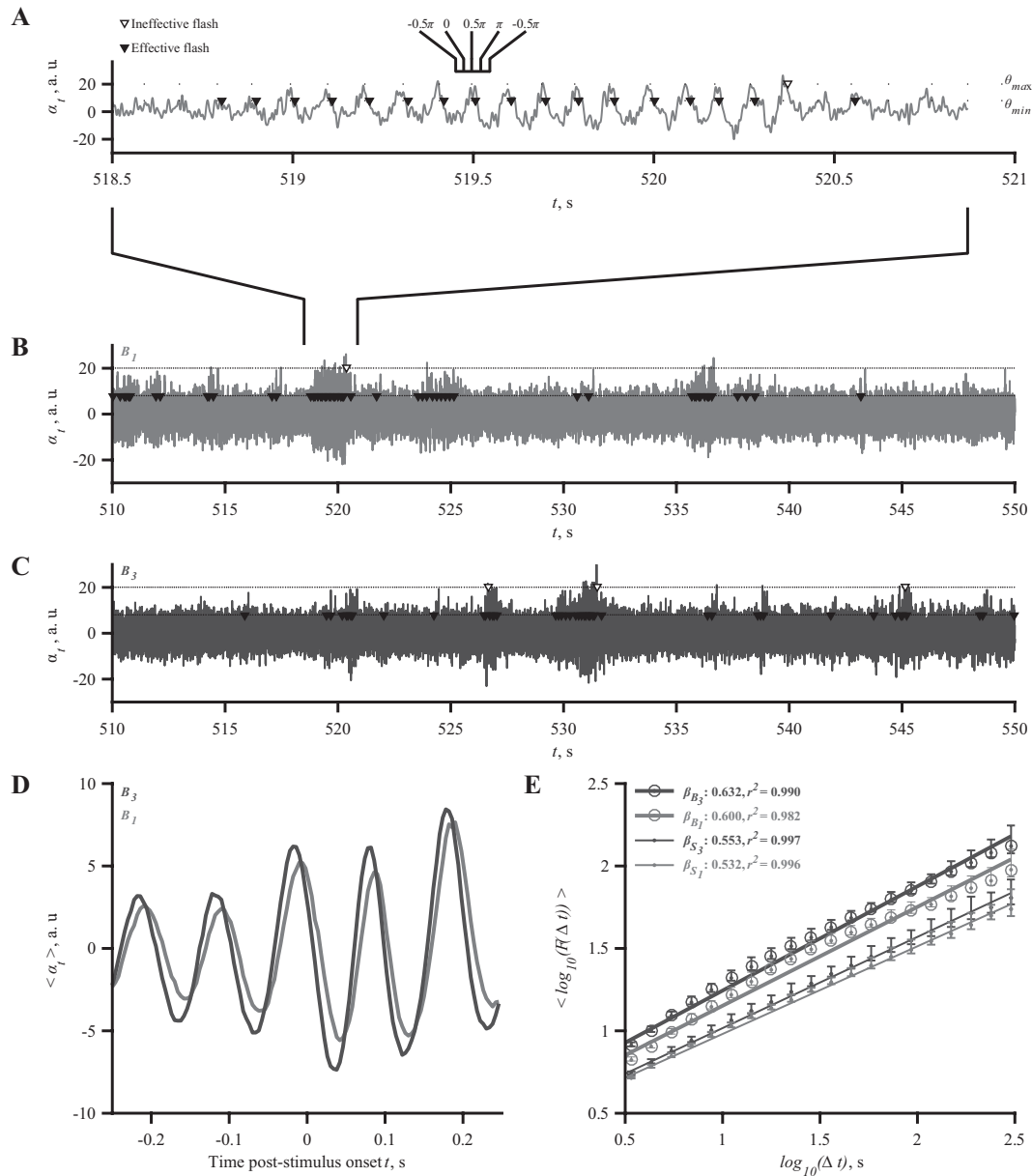


Fig. 1. A simulated noisy amplitude-modulation of a noisy period-modulated 10 Hz harmonic oscillator, with neurofeedback at (A) 0.5π to π (B) eliciting complexes of overlapping flash-evoked deterministic ERP alpha-band waveforms and exponential oscillatory amplitude gain in the O2 recording of alpha-band, α_t , during the first neurofeedback block B_1 . Effective neurofeedback succeeds high-amplitude oscillations not exceeding a physiological maximum, θ_{max} . Composite ERP-oscillatory waveforms trigger flash volleys until the influence of physiological sources, including this exponential gain, rise over a physiological maximum or, fall below a calibrated minimum, θ_{min} , ceasing the contribution of oscillatory gain. Supplementary Materials define this model. (C) Exponential gain is stronger on the third neurofeedback block B_3 . (D) Grand-averaged $\langle \alpha_t \rangle$ time-locked to flashes exhibits both pre- and post-flash differences between neurofeedback blocks as flashes occur in volleys due to partially overlapping ERP complexes and oscillatory gain spindles. Neurofeedback block influences (E) LRTC Detrended Fluctuation Analysis neurofeedback mean exponents (block 3: β_{B_3} > block 1: β_{B_1}) exceeding surrogate data exponents (β_{S_3} and β_{S_1}).

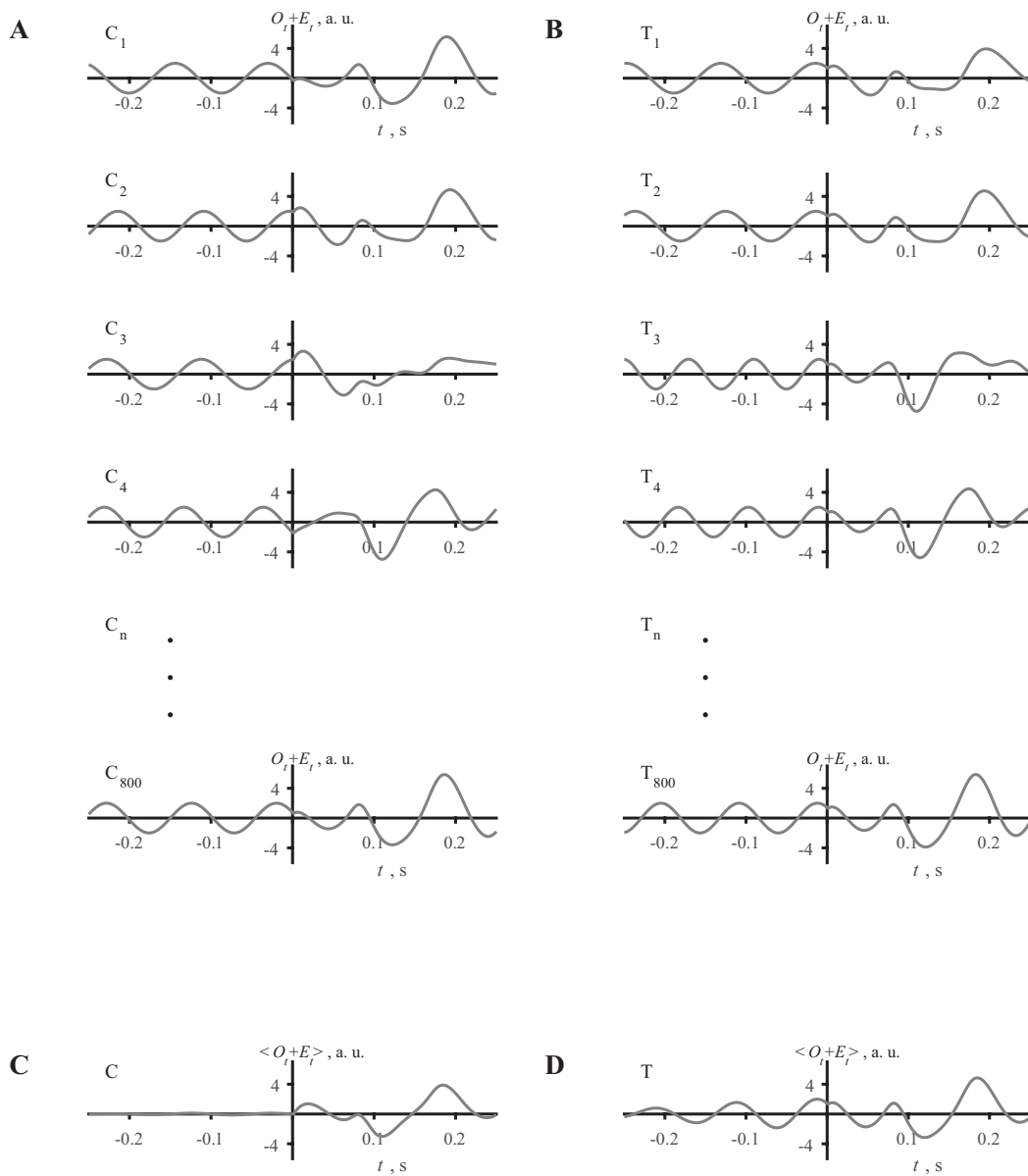


Fig. 2. Phase-locking of the presentation of stimulation to the alpha oscillation can cause constructive averaging influencing the representation of ongoing alpha in the pre-stimulus baseline. Different schematic epochs of alpha-band EEG, each at a different set of discrete time-points, t , contain ongoing oscillations O_t , simplified to $2\sin(2\pi f_t t + \varphi)$, such that $f_t \sim \mathcal{N}(10, 1)$ is constant within an epoch, added to the deterministic ERP, E_t , as detailed in Supplementary Materials, with only one flash-evoked deterministic ERP evoked per epoch. (A) The phase $\varphi \sim \mathcal{N}(0, 1)$ of O_t is broadly inconsistent at corresponding time-points relative to different stimulus onsets in each simulated sham control epoch, C_n , as can often be in counter-phase between epochs. (B) Contrastingly, the phase, $\varphi = \frac{7\pi}{8}$, of O_t , is broadly consistent at corresponding time-points in each simulated neurofeedback treatment epoch, T_n , and very rarely in counter-phase. In line with Zhigalov *et al.*'s Fig. 4, ongoing alpha oscillations are thus less strongly represented in the pre-stimulus baseline of averages of 800 epochs, $\langle O_t + E_t \rangle$, time-locked to (C) control sham epochs C then for (D) treatment neurofeedback epochs T .

1.6. Higher amplitude ongoing alpha in the neurofeedback treatment than sham control baseline

Delivering the neurofeedback flash at a fixed interval from the alpha peak causes ongoing alpha to represent more strongly in the pre-stimulus baseline. That is, the neurofeedback treatment epochs were phase-locked to a particular point in the alpha cycle. On the sham control trials, there was no such phase-locking, alpha cycles appearing in epochs starting at multiple phases with flash onset. As such, in averaging alpha cycles were of very similar phase in all neurofeedback treatment epochs averaged, of differing phase in all sham control epochs averaged. After averaging the alpha waveform phase-locked to the onset is thus apparent in a feedback not sham averages of Zhigalov *et al.*'s Figure 4. As schematised in Fig. 2, this ongoing alpha thus considerably averages-out on sham trials.

1.7. Long-term effects of neurofeedback in the pre-stimulus baseline

This ongoing occipital alpha, phase-locked to the flash, is higher in amplitude on the third neurofeedback block than on other blocks. As simulated in Fig. 1A–C, if flashes and the subsequent alpha augment occurred in volleys, effects of neurofeedback from the flashes beforehand not only influence the post-stimulus ERP but also, more subtly, the pre-stimulus baseline (Fig. 1D). Zhigalov *et al.* adopt the notion that there is an interaction between ongoing neuronal activity and evoked responses rather than their linear summation. A corollary is that ERPs are an amplitude modulation of non-zero mean oscillations in the ongoing EEG (Nikulin *et al.*, 2007).

1.8. Simulations with an alternative micromodel

Rather, higher ERP amplitudes due to occipital alpha contamination phase-locked to flashes support another model: noise and ongoing EEG oscillations summate linearly with an invariant ERP signal to each stimulus of a given class (Ivannikov *et al.*, 2009). Building on this notion of linear summation, in simulations with the micromodel detailed in Supplementary Material, occipital alpha measurements derive from addition of the neurofeedback-modulated deterministic contribution of single-trial flash-evoked ERPs to ongoing alpha oscillations and a neurofeedback flash-evoked gain in the amplitude of those oscillations. Neurofeedback influences simulated power-scaling (Fig. 1E) of LRTCs (Fig. 1B–C) without the inclusion of a critical branching assumption. This micromodel is phenomenological, an illustrative set of mathematical principles offering parameters (Table 1 of Supplementary Material) of a preliminary fit to Zhigalov *et al.*'s data, which could be further optimised. These principles comment on the effects simulated. Whilst critical branching might be considered a mechanism, the observed effects do not warrant the conclusion that these effects influence such a mechanism. These effects cannot reveal the biological mechanisms of the effects and as such, although feasible, the principles of the micromodel offered in Supplementary Material are not specified at a mechanistic level of detail. Rather, as the implementation demonstration-proof that critical branching is not a necessary mechanism, the micromodel reveals effects on the, seemingly critical, power-scaling of LRTCs: This micromodel uses random Gaussian noise generated with a computer algorithm instead of a critical branching process (for a related perspective, please see Botcharova *et al.*, 2015). This micromodel does make unprecedented predictions testable with this dataset. For instance, predictions include that neurofeedback flashes would occur in volleys (Fig. 1A–C).

1.9. Concluding remarks

Zhigalov *et al.*'s findings neither conclusively support a theory of critical branching between neuronal elements nor deny stimulus-evoked ERPs theoretically deterministic

characteristics. In light of the foregoing issues, a further concern is that the clinical potential of this approach to neurofeedback implied remains empirically undetermined.

1.10. Conflict of interest statement

None of the authors have potential conflicts of interest to be disclosed.

References

- Zhigalov, A., Kaplan, A., and Palva, J.M. (2016). "Modulation of critical brain dynamics using closed-loop neurofeedback stimulation.", *Clin. Neurophysiol.*, **127**(8), 2882–2889.
- Palva, J.M., Zhigalov, A., Hirvonen, J., Korhonen, O., and Linkenkaer-Hansen, K. (2013). "Neuronal long-range temporal correlations and avalanche dynamics are correlated with behavioral scaling laws.", *Proc. Nat. Acad. Sci. U.S.A.*, **110**(9), 3585–3590.
- Siebenhühner, F., Lobier, M., Wang, S.H., Palva, S., and Palva, J.M. "Measuring large-scale synchronization with human MEG and EEG: challenges and solutions.". In S. Palva (Ed.) *Multimodal Oscillation-based Connectivity Theory* (Springer International Publishing, 2016), 1–18.
- Korhonen O., Palva S., and Palva J.M. (2014). "Sparse weightings for collapsing inverse solutions to cortical parcellations optimize M/EEG source reconstruction accuracy.", *J. Neurosci. Methods*, **226**, 147–160.
- Näätänen, R., and Winkler, I. (1999). "The concept of auditory stimulus representation in cognitive neuroscience.", *Psychol. Bull.*, **125**(6), 826–859.
- Campbell, T., Winkler, I., and Kujala T. (2007). "N1 and the mismatch negativity are spatiotemporally distinct ERP components: Disruption of immediate memory by auditory distraction can be related to N1.", *Psychophysiology*, **44**(4), 530–540.
- Ruby, P., Caclin, A., Boulet, S., Delpuech, C., and Morlet, D. (2013). "Odd sound processing in the sleeping brain.", *J. Cogn. Neurosci.*, **20**(2), 296–311.
- Morlet, D., and Fischer, C. (2014). "MMN and novelty P3 in coma and other altered states of consciousness: a review.", *Brain Topogr.*, **27**(4), 467–479.
- Campbell, T.A. (2015). "A theory of attentional modulations of the supratemporal generation of the auditory mismatch negativity", *Front Hum. Neurosci.*, **8**:1065 .
- Wiens, S., Szychowska, M., and Nilsson, M.E. (2016). "Visual task demands and the auditory mismatch negativity: an empirical study and a meta-analysis", *PLoS One*, **11**(1):e0146567 .
- Lightfoot, G., and Kennedy, V. (2006). "Cortical electric response audiometry hearing threshold estimation: accuracy, speed, and the effects of stimulus presentation features.", *Ear Hear.*, **27**(5), 443–456.
- Busch N.A., Dubois, J., and VanRullen R. (2009). "The phase of ongoing EEG oscillations predicts visual perception.", *J. Neurosci.*, **29**(24), 7869–7876.
- Lyyra, P., Wikgren, J., and Astikainen P. (2010). "Event-related potentials reveal rapid registration of features of infrequent changes during change blindness.", *Behav. Brain Func.*, **6**(12) .
- Sysoeva O.V., Lange E.B., Sorokin, A.B., and Campbell, T. (2015). "From pre-attentive processes to durable representation: An ERP index of visual distraction.", *Int. J. Psychophysiol.*, **95**(3), 310–321.
- Ivannikov, A., Kalyakin, I., Hämäläinen, J., Leppänen, P.H.T., Ristaniemi, T., Lyytinen, H., *et al.* (2009). "ERP denoising in multichannel EEG data using contrasts between signal and noise subspaces.", *J. Neurosci. Methods*, **180**(2), 340–351.
- Yantis, S., and Hillstrom, A.P. (1994). "Stimulus-driven attentional capture: evidence from equiluminant visual objects.", *J. Exp. Psychol. Hum. Perc. Perf.*, **20**(1), 95–107.
- Nikulin, V.V., Linkenkaer-Hansen, K., Nolte, G., Lemm, S., Müller, K.R., Ilmoniemi, R.J., *et al.* (2007). "A novel mechanism for evoked responses in the human brain.", *Eur. J. Neurosci.*, **25**(10), 3146–3154.
- Botcharova, M., Berthouze, L., Brookes, M.J., Barnes, G.R., and Farmer, S.F. (2015). "Resting state MEG oscillations show long-range temporal correlations of phase synchrony that break down during finger movement.", *Front. Physiology*, **6**:183 .

Supplementary Material

Does occipital alpha neurofeedback require critical branching to causally influence LTRCs and deterministic ERPs?

Tom Campbell

*Institute for Behavioural Sciences, University of Helsinki,
00014 Helsinki, Finland.
tom.campbell@helsinki.fi*

2. Supplementary Material

2.1. The simulation model

The simulation model (Eq. 1) assumes that measures of an EEG alpha-band time-series, α_t , at discrete time-points t comprise the addition of ongoing neuronal oscillations, O_t , to the deterministic contribution to single-trial flash-evoked ERPs, E_t , to neurofeedback flash-evoked gain in the amplitude of those oscillations, G_t , all of which are contaminated by the linear addition of additive Gaussian white noise $n_t \sim \mathcal{N}(0, v)$, v being the variance of zero-mean noise controlled by multiple physiological and non-physiological factors. These noise factors correlate neither with these contributions to alpha oscillation generation nor with ERPs:

$$H = O_t + E_t + G_t + n_t \quad (1)$$

In the follows, each of the first three terms in this simulation model are introduced, leading into the basic dynamics and operation of the model. The principles introduced foreshadow how the simulation model comments upon the averaged data and the power-scaling of long-range temporal correlations (LTRCs) in the continuous alpha-band data of [Zhigalov et al. \(2016\)](#). The discussion now turns to the first term of the model.

2.2. Ongoing oscillations

Ongoing oscillations, O_t , are amplitude modulations of a harmonic oscillator, $\sin(2\pi ft + \pi)$, of a frequency f (Eq. 2) that the influence of thalamic and neocortical pacemakers on occipital neuronal elements determines, for instance via shunting inhibition ([Scheeringa et al., 2011](#)). The period of those ongoing oscillations, determined by multiple factors, has a Gaussian distribution such that $f_t \sim \mathcal{N}(10, 1)$ with a mean of 10 Hz and a variance set to 1 for definiteness. Multiple physiological factors related to amplitude-modulation of ongoing alpha-band oscillations produce multiplicative zero-mean Gaussian white noise $m_t \sim \mathcal{N}(0, u)$ with variance u . In turn, a smoothing of the absolute value of this physiological multiplicative noise, p_t , (Eq. 3) amplitude-modulates each oscillation. This process produces a smooth random modulation of the ongoing oscillation that is typically low but occasionally high (Eq. 2).

$$O_t = p_t \sin(2\pi ft + \pi) \quad (2)$$

$$p_t = \frac{\sum_{t-9}^t |m_t|}{10} \quad (3)$$

The neuronal underpinnings of long-range temporal correlations in the alpha-band could be amongst the multiple physiological factors that determine $m_t \sim \mathcal{N}(0, u)$. However, the presence of strong power-scaling of LRTCs in resting-state alpha amplitude modulation without neurofeedback or stimulation, is unnecessary as ground condition: Without such strong power-scaling initially present, it is possible to simulate the influence of neurofeedback flashes on averaged waveforms time-locked to those flashes. It is also possible to simulate how neurofeedback causes power-scaling of LRTCs as well as influencing that power-scaling. Here, $m_t \sim \mathcal{N}(0, u)$, rather, simplifies to a random matrix of Gaussian white noise with the constraint that values are identical within an oscillation but do not predict the values on subsequent oscillations. However, smoothing (Eq. 3) does render the amplitude modulation of oscillations predictable on the timescale of 10 oscillations, ca. 1 s. The resulting ongoing oscillations, O_t , contaminated by additive noise unrelated to alpha oscillations, $n_t \sim \mathcal{N}(0, v)$, suffice for occasional alpha peaks of α_t , at the peak of O_t , to exceed an individually calibrated threshold θ_{min} even without neurofeedback stimulation.

2.3. Deterministic ERPs

Each flash then evokes the theoretically deterministic contribution to a single-trial ERP waveform with, accordingly, an invariant time-course of these deterministic ERPs. Turning to this second E_t term of the model, these deterministic ERP waveforms are derived on an empirical basis from Fig. 4 of Zhigalov *et al.* (2016). These deterministic ERP waveforms follow the time-course of the grand-averaged ERP to the first set of sham blocks grand-averaged O2 ERP from 0 to 250 msec post-stimulus. In this simulation, in response to each flash, each deterministic ERP, w , thus lasts 250 msec. With deterministic ERPs elicited by flashes in rapid succession, deterministic ERPs elicited by a single prior flash or several prior flashes can overlap the deterministic ERP to the most recent flash. When such overlap of deterministic ERPs occurs, deterministic ERPs add together constructively in the ongoing time-series, w_t . There is an emergent property of such overlapping deterministic ERPs with inter-flash intervals near to 100 msec, as would occur with stimuli presented 12.5 msec after the peak of consecutive alpha oscillations. That is, the refracted P1 to the most recent flash adds to the refracted P2 that the preceding stimulus elicited. Flashes, which are phase-locked to consecutive alpha oscillations that are relatively stable in period, which $f_t \sim \mathcal{N}(10, 1)$ engenders in (Eq. 2), thus give rise to complexes of overlapping deterministic ERPs. The schedule of stimulation evokes these deterministic ERPs. The time-series, E_t , thus contains these deterministic ERPs and their complexes. E_t is otherwise set to zero without a recent flash.

While the time-course of deterministic ERPs is invariant, exposure to neurofeedback influences the amplitude of the deterministic generation of the componentry of those ERPs. Relatively longstanding factors caused by exposure to neurofeedback – for instance, drowsiness, learning, and changes in sustained or selective attention to flashes – lead to increases in the blockwise strength coefficient, s , as a function of neurofeedback block number. While these factors may operate gradually over a matter of minutes, the model emulates their contribution blockwise. This blockwise increment in strength, s , augments the amplitude of the contribution of the componentry of deterministic ERPs w_t to E_t (Eq. 4). Accordingly, the amplitude of each deterministic ERP w weighted by s is higher during later, neurofeedback blocks, during which an assumption is that neurofeedback has more influence:

$$E_t = sw_t \quad (4)$$

2.4. Neurofeedback-related exponential occipital oscillatory alpha amplitude gain

On a shorter-term timescale, for each effective flash, a neurofeedback flash-related gain in the amplitude of the subsequent oscillation, G_t , increases exponentially according to the number of flashes occurring during preceding consecutive alpha peaks (Eq. 5). It is to this G_t term of the simulation model that the discussion now turns. The exponent of this gain is the number of preceding effective flashes, l , on consecutive oscillations weighted by a gain coefficient, g , determining the increasing potency of each such consecutive flash to increment gain on the next oscillation. Accordingly, independent of influences on deterministic ERPs, relatively longstanding factors caused by exposure to neurofeedback – as might include drowsiness, learning, and changes in sustained or selective attention to flashes – lead to a modulation of this gain blockwise. While these factors may operate gradually over a matter of minutes, the model implements their contribution blockwise. Increments in a blockwise coefficient, b , thus also increase the flash-related exponential gain, G_t , in the next alpha oscillation as neurofeedback becomes more effective from block-to-block:

$$G_t = be^{gl} \sin(2\pi ft + \pi) \quad (5)$$

2.5. Model dynamics and operation

Having introduced the terms of the model, this discussion turns to the models dynamics and operation. Neurofeedback flashes occur between 0 to π of oscillations of $-\pi$ to π radians of the oscillation, $\sin(2\pi ft + \pi)$, i.e., during the peak rather than the trough of an alpha oscillation (Fig. 1A), as in Zhigalov *et al.* (2016) neurofeedback blocks. Consequently, there is a reduction in the occipital haemodynamic response to such visual stimuli (Scheeringa *et al.*, 2011). Whilst limiting the occipital haemodynamic response to stimulation, an assumption is that such flashes during alpha peaks (Fig. 1A) tend to increase occipital alpha power on the subsequent oscillation. As specified in Eq. 5, for each such effective flash, l thus increments by 1. Each such effective neurofeedback flash thus increases gain, G_t , elevating the probability that the peak amplitude of the next oscillation of α_t will exceed a calibrated threshold, θ_{min} , at the peak of O_t . In this way, flashes occur in volleys triggered by alpha spindles (Fig. 1A–C) rising in amplitude over this threshold according to an exponential tendency. When α_t reaches a primarily physiologically determined maximum, θ_{max} , l returns to 0 such that the gain G_t is also 0 for the subsequent oscillation. The triggered neurofeedback flash is thus ineffective. In turn, that gain does not contribute to the amplitude of that oscillation, tending to bring the reciprocal neurofeedback between a spindle of oscillatory gain, complexes of deterministic ERPs, and a volley of flashes to a gradual closure. These dynamics apparent for neurofeedback block 1 in Fig. 1B are more prominent for neurofeedback block 3 in Fig. 1C. Whilst increases in oscillatory gain occur on the cycle following an effective flash, each flash elicits a deterministic ERP – whether effective or ineffective. In the simulated dynamics of α_t , such ineffective flashes occur (Fig. 1B–C) more frequently on the later block 3.

Multiple factors producing physiological noise, p_t , related to the strength of amplitude modulation or potentials from multiple unrelated sources of physiological or non-physiological noise n_t , can together occasionally cause α_t at the peak of the oscillation to fall below the pre-calibrated minimum, θ_{min} , for triggering a new flash. α_t can fall below this threshold even when there is an increase in oscillatory gain, G_t . Falling below this θ_{min} threshold, at the peak of O_t , also brings the reciprocal interaction between neurofeedback flashes, the spindle of oscillatory gain, and deterministic ERPs to a closure. Accordingly, as simulated in Fig. 1B–C, volleys of neurofeedback flashes can thus end without an ineffective flash.

2.6. Preliminary parameter fits for simulating Zhigalov et al.'s dataset

A variety of dynamics is available within the model's parameter space, and these parameters might be tuned to fit individual data. Having introduced the terms, dynamics and operation of the model, the discussion now turns to the simulation of Zhigalov et al. (2016)'s data. Table 1 overleaf details parameters for the simulation depicted in Fig. 1 that centres on fitting the grand-averaged O2 waveform from neurofeedback blocks 1 and 3 (Zhigalov et al., 2016). In this simulation, 9 neurofeedback blocks 1 and 3 of 20 minutes, 180 minutes of α_t , these parameters emulate alpha-band data α_t at O2 for 9 representative participants.

500 msec epochs of α_t with a 250 msec pre-stimulus baseline for each participant were binned separately for the first and third neurofeedback blocks collapsing across epochs time-locked to effective and ineffective flashes, averaged, and these simulated individual weighted-average waveforms then grand-averaged in an unweighted manner. The primary goal of the simulation was to model averaged data from such simulated individual participants for which an actual grand-averaged O2 waveform was available (Fig. 4 of Zhigalov et al., 2016). The simulated grand-averaged waveform of amplitudes $\langle \alpha_t \rangle$ as a function of times post-stimulus onset met this goal to the extent depicted in Fig. 1D.

2.7. Simulated ascending alpha gain spindles and deterministic ERP

Spindles of exponentially ascending alpha gain, additive to deterministic ERP complexes, and the consequent reciprocal volleys of neurofeedback flashes became more prevalent on the later blocks, when neurofeedback becomes more influential. In turn, the averaged simulated alpha-band amplitude $\langle \alpha_t \rangle$ (Fig. 1D) increased from early to later neurofeedback blocks. This increase occurs not only after the stimulus but also before the stimulus given that neurofeedback flashes in volleys also occur in the pre-stimulus baseline.

2.8. Simulated LRTCs power-scaling for neurofeedback compared to surrogate and rest blocks

In this simulation, stimulation volleys, deterministic ERP complexes, and spindles of increasing oscillatory alpha gain in α_t impact the power-scaling of LRTCs (Fig. 1E), as Zhigalov et al. (2016) reveal. Detrended Fluctuation Analysis, variously known as DFA (Peng et al., 1994; Hardstone et al., 2012) yields exponents for simulated data from neurofeedback blocks 1 and 3. Respectively, these exponents have a mean β_{B_1} , 0.600, 95% Confidence Intervals (CI) [0.585, 0.641], and a mean β_{B_3} , 0.633, 95% CI [0.621, 0.665]. These mean exponents are higher than the corresponding surrogate data mean exponents. That is, for the counterpart phase-randomised data, these surrogate exponents have a lower mean β_{S_1} , 0.532, 95% CI [0.519, 0.569], and mean β_{S_3} , 0.554, 95% CI [0.537, 0.600]. For neither set of neurofeedback blocks does the range of power-scaling exponents overlap that of the corresponding surrogate data. These neurofeedback block exponents are higher because the surrogate data do not contain the temporal structure emergent in the simulated neurofeedback blocks.

There is further support for this notion that deterministic ERP complexes and spindles of increasing oscillatory alpha gain introduce the temporal structure reflected by the LRTC: These mean power-scaling exponents of the phase-randomised surrogate data reside more within the lower range of the simulated resting-state α_t mean exponent, β_R , 0.503, 95% CI [0.483, 0.560], without stimulation. For neither neurofeedback block does the range of power-scaling exponents overlap that for the corresponding surrogate data. Given stimulation does not occur in this resting-state control, simulation of such resting-state data relies on a subset of the terms of Eq. 1. These terms include ongoing alpha-band oscillations O_t , into which the smoothing of

Table 1. Simulation model parameters and values.

Parameter	Symbol	Value
Variance of alpha oscillation-unrelated additive Gaussian white noise, such that $n_t \sim \mathcal{N}(0, v)$	v	4.00
Variance of alpha oscillation-related multiplicative Gaussian white noise, such that $m_t \sim \mathcal{N}(0, u)$	u	2.31
Mean frequency of the harmonic alpha oscillator, such that $f_t \sim \mathcal{N}(10, 1)$	f	1.00×10
Calibrated threshold for a neurofeedback flash	θ_{min}	8.04
Physiological maximum of EEG	θ_{max}	2.01×10
Neurofeedback block 1 blockwise deterministic ERP strength coefficient modulation	s_{B_1}	9.10×10^{-1}
Neurofeedback block 3 blockwise deterministic ERP strength coefficient modulation	s_{B_3}	1.19
Neurofeedback increase in occipital oscillatory alpha gain exponent per effective flash	g	6.6×10^{-2}
Neurofeedback block 1 blockwise occipital oscillatory alpha gain modulation	b_{B_1}	1.00
Neurofeedback block 3 blockwise occipital oscillatory alpha gain modulation	b_{B_3}	2.70

Eq. 3 introduces autocorrelational temporal structure. That resting-state data also include noise, n_t , unrelated to those oscillations. This resting-state control data, regardless of said smoothing, exhibits the power-scaling exponent of a near-random process. The higher range of neurofeedback exponents thus overlapped less with the range of the power-scaling surrogate data exponents than the near-random range of simulated resting-state exponents overlapped with that of surrogated data exponents.

Parenthetically, in departure from [Palva et al. \(2013\)](#) and [Zhigalov et al.](#), the DFA convention of [Hardstone et al. \(2012\)](#)'s Neurophysiological Biomarker Toolbox

(<https://www.nbtwiki.net/>) utilised for the simulation data here yielded positive rather than negative y -values for each time window on the log-log plot denoting the (mean) log of the fluctuation function $< \log_{10}(F(\Delta t)) >$. Exponents, the positive slopes of the log-log plot in Fig. 1E for the simulation data, are thus loosely comparable with Zhigalov *et al.* (2016)'s similarly positive slopes for actual data on such log-log plots.

This influence of neurofeedback block on the power-scaling of LRTCs, shown comparing simulated neurofeedback blocks with surrogate data (Fig. 1E), need not require critical branching, as Zhigalov *et al.* (2016) imply. Rather, this simulated influence of neurofeedback on LRTCs relies on a long-term dynamics emergent from flash-elicited complexes of deterministic ERPs and accumulating exponential oscillatory alpha gain spindles due to closed-loop flash neurofeedback stimulation during consecutive alpha oscillation peaks. The micro-behaviours (Fig. 1A) produced by mechanisms of exponential occipital oscillatory alpha gain and deterministic ERP generation have macroscale consequences influencing the power-scaling of LRTCs (Fig. 1B–C, 1E).

2.9. Simulating block-to-block changes in LRTC power-scaling

This discussion now turns to the block-to-block change in the influence of neurofeedback block on LRTCs. As apparent in the slopes of Fig. 1E, this change increases the power-scaling exponent of LRTCs exponents from β_{B_1} to β_{B_3} : In Zhigalov *et al.*'s data, the mean β_{B_1} (0.695) on the neurofeedback block 1 is near-identical to the mean exponent from the corresponding first sham block. By contrast, mean β_{B_3} is higher on neurofeedback block 3 (0.671) than on the third sham block (0.647). As an aside, it is worth considering that significant changes in mean β , denoting power-scaling of LRTCs, are visible in the actual data (Zhigalov *et al.*, 2016, Fig. 1B) from the first (0.695) to third sham blocks (0.647). This block-to-block change in temporal structure is neither attributable to neurofeedback nor to changes in the influence of deterministic ERPs nor to oscillatory alpha gain.

The model's assumptions, centred on neurofeedback, are agnostic to these relatively longstanding confounding changes, apparent in the sham, which led to an attenuation in the power-scaling exponents LRTCs from the first to the third neurofeedback block in the actual data. Candidate explanations of these longstanding changes might include the consequences of continued mere exposure to flashes on temporal structure rather than neurofeedback effects per se.

Given the actual β_{B_1} was near-identical on the first set of neurofeedback and the first set sham blocks, the empirically motivated simulation control, circumventing the confounding influence of these longstanding changes, should concern an unfounded effect of neurofeedback block ($\beta_{B_3} > \beta_{B_1}$). This effect should, for definiteness, be as strong as the difference between the actual sham LRTC exponent on block 3 (mean: 0.647) and the actual neurofeedback LRTC exponent on block 3 (mean: 0.671), i.e., 0.024. The model surpasses this criterion (Fig. 1E), with an increase in mean LRTCs exponents for neurofeedback blocks 1 and 3 of 0.033. That is, the simulation model thus takes a tenable stance on how neurofeedback on the later third block boosts the power-scaling exponents of LRTCs in a way that the neurofeedback on the first block does not.

On balance, neurofeedback has more influence on the later block 3, as apparent in the averaged waveforms (Fig. 1D), because of a faster rise in exponential oscillatory alpha gain, as well as an additive increase in the amplitude of deterministic ERP componentry visible in Figs. 1C–D. On the later block, the simulations results reveal spindles of oscillatory gain superposed upon higher-amplitude deterministic ERPs, yielding volleys of reciprocal neurofeedback stimulation with a higher proportion of ineffective flashes in those volleys due to α_t more regularly exceeding the physiological maximum, θ_{max} . When that maximum is thus exceeded, the ineffectiveness of those flashes to yield an increase in alpha only occasionally brings a neurofeedback stim-

ulation reciprocal valley to an immediate closure. This ineffective flash still elicits a deterministic ERP, which is higher in amplitude than on the first block. That deterministic ERP, insensitive to whether the flash was effective or ineffective in producing an increase in oscillatory gain, reduces the chances of the next few peaks of α_t , at the peaks of O_t , falling below the calibrated minimum θ_{min} for triggering a flash.

Just as when comparing neurofeedback blocks to surrogate data, considering neurofeedback blocks 1 and 3, the block-to-block change in power-scaling of LRTCs (Fig. 1E) need not require critical branching. Rather, the blockwise changes in long-term dynamics are emergent from several factors operating during the third block of closed-loop flash neurofeedback stimulation. These factors are flash-elicited complexes of higher-amplitude deterministic ERPs and the more rapidly accumulating exponential oscillatory alpha gain spindles during closed-loop stimulation upon consecutive alpha oscillations up to a physiological maximum.

2.10. Concluding remarks

In assessment, the modelling process simulates aspects of alpha-band continuous EEG (Fig. 1A–C), alongside power-scaling of LRTCs (Fig. 1E), as well as the averaged data from neurofeedback blocks as depicted in Fig. 1D. These averages resemble Zhigalov *et al.*'s grand-averaged waveforms. This simulation not only relies upon flash-related exponential increases in oscillatory alpha gain but also relies upon deterministic ERPs (Ivannikov *et al.*, 2009) that add constructively into complexes.

References

- Zhigalov, A., Kaplan, A., and Palva, J.M. (2016). "Modulation of critical brain dynamics using closed-loop neurofeedback stimulation.", *Clin. Neurophysiol.*, **127**(8), 2882–2889.
- Scheeringa, R., Mazaheri, A., Bojak, I., Norris, D.G., and Kleinschmidt, A. (2011). "Modulation of visually evoked cortical fMRI responses by phase of ongoing occipital alpha oscillations.", *J. Neurosci.*, **31**(10), 3813–3820.
- Peng, C.-K., Buldyrev, S.V., Havlin, S., Simons, M., Stanley, H.E., and Goldberger, A.L. (1994). "Mosaic organization of DNA nucleotides.", *Phys. Rev. E*, **49**(2), 1685–1689.
- Hardstone, R., Poil, S.-S., Schiavone, G., Jansen, R., Nikulin, V.V., Mansvelder, H.D., *et al.* (2012). "Detrended fluctuation analysis: A scale-free view on neuronal oscillations.", *Front. Physiology*, **3**:450.
- Palva, J.M., Zhigalov, A., Hirvonen, J., Korhonen, O., and Linkenkaer-Hansen, K. (2013). "Neuronal long-range temporal correlations and avalanche dynamics are correlated with behavioral scaling laws.", *Proc. Nat. Acad. Sci. U.S.A.*, **110**(9), 3585–3590.
- Ivannikov, A., Kalyakin, I., Hämäläinen, J., Leppänen, P.H.T., Ristaniemi, T., Lyytinen, H., *et al.* (2009). "ERP denoising in multichannel EEG data using contrasts between signal and noise subspaces.", *J. Neurosci. Methods*, **180**(2), 340–351.